PATHOLOGY OF ADENOIDS AND ADENOID TUBERCULOSIS.[1]

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Although the clinical conditions associated with adenoids and enlarged tonsils are now widely recognized, the pathology of these overgrowths is not fully understood. In the present paper I have attempted to summarize some of the views recently advanced concerning the pathology of these tissues, especially as to their relation to tuberculosis. The subject has been investigated in connection with the study of a series of adenoids primarily with a view to determining the incidence and significance of tuberculosis in these tissues. In these examinations evidence of tuberculosis has been the main object of search, but I have taken the opportunity to note the general histological features which have been the subject of comment by other observers.

The material was obtained from the Nose, Throat, and Ear Department of the Royal Victoria Hospital. Seventy-five adenoids were examined histologically, and in 12 cases this was supplemented by the inoculation of guinea-pigs. In many cases the faucial tonsils were also carefully examined. The study does not represent a consecutive group of cases, as too much material was available and the work was interrupted on several occasions; on the other hand, it is not selective, except in regard to a few of the cases used for inoculation. The examination has been thorough in each case, and I feel that the findings represent a fairly accurate estimate of the incidence of tuberculosis in the cases studied.

Cases of Adenoid Tuberculosis. In 5 cases histological evidence of tuberculosis was found; in 4 of these the disease was clinically primary in the adenoid, in the fifth the adenoid was probably involved secondarily to a pulmonary focus, and in this case the faucial tonsils were also affected. The percentage of primary adenoid tuberculosis found was thus 5.3 per cent. The notes of these cases, together with the one of secondary involvement, are as follows:

Case I.—Male, aged nine years; adenoid removed February, 1905.

Case II.—Female, aged eighteen years; adenoid removed November, 1905. These 2 cases are very similar as to history. Both gave a family history of tuberculosis, but showed no physical signs of tuberculosis either before or after operation (report from family physician, October, 1906). Sections of the adenoids showed typical tubercles with giant cells and caseation (Figs. 1 and 2). Tubercle bacilli were not demonstrated.

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[1] Read at a meeting of the Montreal Medico-Chirurgical Society, February 15, 1907.

CASE III.—Male, aged seventeen years; adenoid removed November, 1905. A history of family tuberculosis, and of hip-joint

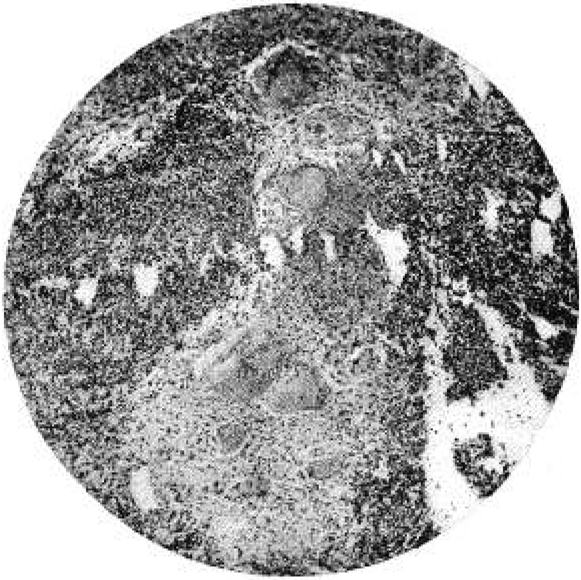

FIG. 1.—Adenoid tuberculosis (Case II). Confluent tubercles with abundant giant cells and caseation. (No. 3 objective.)

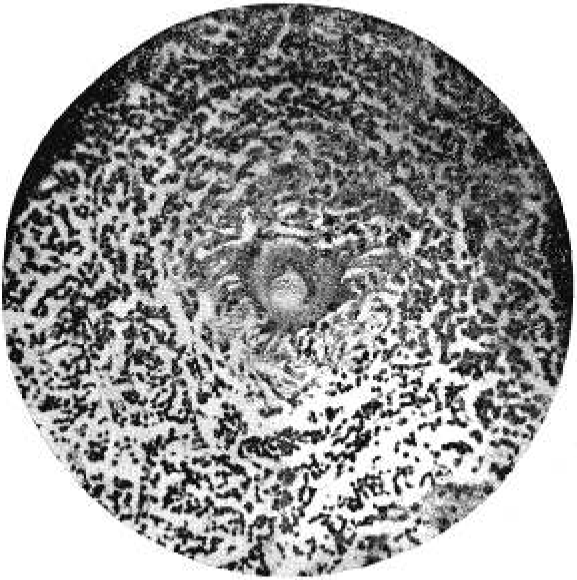

FIG. 2 —Adenoid tuberculosis (Case II). Central giant cell surrounded by a zone of epithelioid cells showing defective staining reaction. At the lower part the edge of a neighboring tubercle is seen. (No. 6 objective.)

disease in childhood at age of six years (healed). At present in rubust health and free from signs of pulmonary tuberculosis—before and after operation (examined October, 1906). Sections of adenoid showed typical tubercles (Fig. 3).

CASE IV.—Female, aged nineteen years; adenoid removed January, 1906. Caseous gland at the angle of the jaw on the left side; otherwise free from signs of tuberculosis, and in robust health. Adenoid showed definite tubercles (Fig. 4), but the tonsils showed no sign of involvement. Subsequent history beyond a few weeks is unavailable, but at that time the patient showed no sign of pulmonary involvement.

In these 4 cases the diagnosis of *primary adenoid tuberculosis* has been made (on clinical evidence).

CASE V.—Male, aged twenty-six years; adenoid removed March, 1906. Had a large mass of caseous glands in the left cervical region. Though there were few physical signs of lung involvement, he had a marked tracheitis and slight elevation of temperature. After operation he developed active pulmonary tuberculosis. The adenoid and tonsils both showed tubercles. Diagnosis—secondary adenoid and tonsillar tuberculosis.

SUMMARY OF INOCULATION EXPERIMENTS. The number of guinea-pigs inoculated was 12; 5 died of sepsis; of the remaining 7, 2 were inoculated with adenoid showing histological tuberculosis. No guinea-pig developed signs of tuberculosis. It seems likely that either the virulence of the infection was not sufficient to overcome the guinea-pig's resistance, possibly an obsolete tuberculosis, or that since the foci were discrete and scattered a non-tuberculous portion was inoculated.

THE PATHOLOGY OF ADENOIDS. In discussing the pathology of the adenoid and enlarged tonsils the features which I wish to emphasize are those bearing on the interrelation of the tonsillar tissues and the general health and well-being of the individual: (1) The causes of tonsillar enlargement, and (2) what is known of the function of these tissues which, under certain conditions, may become important channels of infection.

Attention must first be drawn to the fact that the amount of lymphoid tissue normally present in the pharynx is extremely small and analogous to the lymphoid tissue of the small intestine, Peyer's patches. As Bosworth[2] has pointed out, the conventional tonsil of anatomists is really an hypertrophied one and no more normal than an enlarged lymph node elsewhere. On this same ground Jonathan Wright[3] objects to the use of the term "function" in connection with these tissues, for, he maintains, this implies a normal condition of the organ or tissue studied. Of course, most of the tonsils studied by various observers were probably hypertrophied beyond even the degree popularly considered normal; in the present series all were hypertrophied. What modification this may cause in their function is yet to be determined.

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[2] Trans. Amer. Laryng. Assoc., 1901.

[3] New York Med. Jour., 1900.

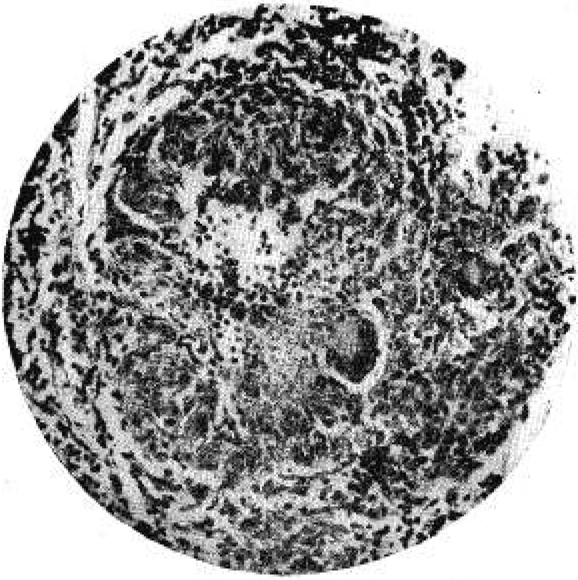

FIG. 3.—Adenoid tuberculosis (Case III). Giant cell and epithelioid proliferation, caseation, and infiltration of the central portion. (No. 6 objective.)

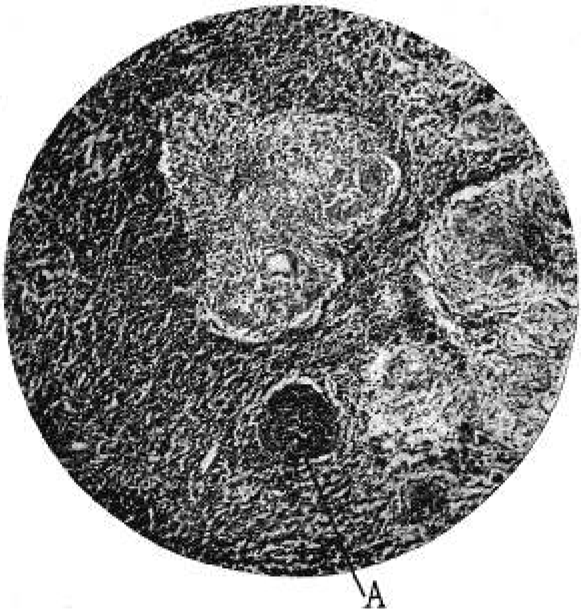

FIG. 4—Adenoid tuberculosis (Case IV). *A*, lymph follicle or germinal centre. The paler areas are tubercles with early caseation. (No. 3 objective.)

ADENOID AND TONSILLAR ENLARGEMENTS. *Histology.* The microscopic appearance of an adenoid or an enlarged tonsil is that of a true hyperplasia, the lymphoid cells being numerically increased. The amount of connective tissue varies to a considerable extent and does not seem to bear any relation to the age of the patient.

Fibrosis was marked in 30 cases of the present series; it was slight or absent in 45 cases. The majority of the cases with fibrosis occurred in the first decade, but marked cases were found in the older subjects. McBride and Turner[4] suggested that the fibrosis frequently seen in adenoids might become a cause of atrophy, but this has not been demonstrated. As Nicholl and Lartigau[5] point out, adenoids which show more or less marked atrophy often lack fibrosis, while others, in which fibrosis is marked, tend to persist. As to the cause of atrophy we know very little, but from the number of adenoids met with in adults, we now know that it is not so constant as was formerly thought.

McBride and Turner[6] point out that atrophy is often inferred from the relief of symptoms as the patient gets older, but that this relief is due rather to the growth of the pharynx making the obstruction relatively less marked than to any true atrophy of the adenoid. It must be emphasized that the most lamentable sequels of adenoids, such as faulty nasal development and chronic nasal or Eustachian catarrh, occur early, during the period of active growth, and that often the adenoid may atrophy only after the harm is done.

The epithelium covering the adenoid is generally columnar, but it is often of transitional or squamous type. In the present series the prevailing type was columnar in 36 cases, squamous in 29, and about evenly mixed in 10 cases. A striking feature of the covering is a rarefaction and lymphoid infiltration of the epithelium. This seems to be constant in the crypts, and very common on the surface as well. This condition has long been commented on and is so extreme at times as to suggest a complete loss of covering. On careful examination, however, the remains of the epithelial cells and the basement membrane can be made out. This change makes the deeper tissue more accessible to invasion by the loss of the mechanical and other protective influences which a healthy mucosa afford. Ewing[7] seems to regard this as an invasion on the part of the lymphoid tissue. Some observers have regarded this condition of the mucosa as evidence of an epithelial origin of lymphocytes. In my opinion it represents merely a chronic inflammatory change in the mucosa, the generative activity of the epithelial cells being directed toward repair rather than to the formation of lymphocytes. A somewhat similar condition is to be seen in the mucosa of polypoidal turbinate bodies. Fig. 5 shows an extreme grade of infiltration of the mucosa and a tuberculous area immediately beneath it, indicating a successful penetration by the tubercle bacilli.

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[4] Edin. Med. Jour., 1897, i.

[5] AMER. JOUR. MED. SCI., 1902.

[6] Edin. Med. Jour., 1897, i.

[7] New York Med. Jour., 1897.

THE CAUSES OF ADENOID AND TONSILLAR ENLARGEMENTS. The causes of the lymphoid hyperplasia may be divided into two groups: (1) Constitutional, or predisposing, and (2) local, or exciting. Of these, the predisposing causes are regarded as much the more important.

As regards the frequency of adenoids in Montreal, Birkett[8] in 10,000 cases seen in private practice, found 1605 with adenoids, or 16 per cent. Of course, this percentage is of persons with nose, throat, or ear symptoms. Among persons otherwise healthy Meyer[9] found that of 1000 cases 1 per cent. had adenoids, and Chappel[10] found adenoids in 3 per cent. of 2000 New York school children examined. Sex does not appear to exert any influence.

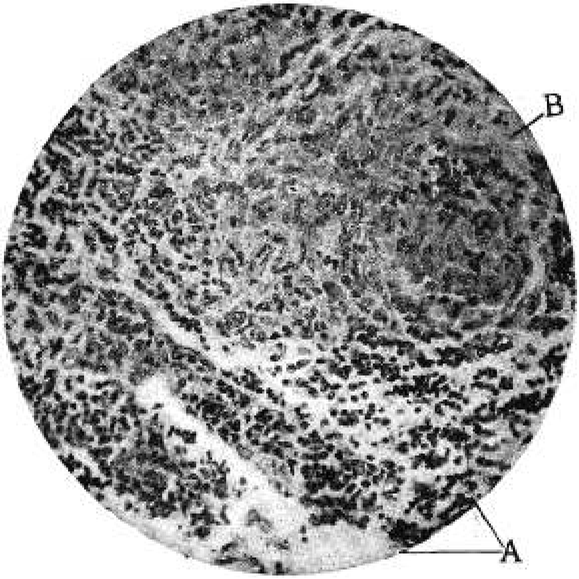

FIG. 5.—Adenoid tuberculosis (Case II). *A*, marked infiltration of the mucosa *B*, tubercle with marked epithelioid proliferation and beginning caseation. (No. 6 objective.)

The age of occurrence in the majority of cases is between the third and fifteenth year. For a short period the greatest number of cases in Birkett's series occurred between the third and fifth year. They are frequently met with up to the twentieth year, and occasionally much later. Cases have been reported in which the condition was found at, or shortly after, birth, but this must be rather rare. Nicholl and Lartigau, in 100 cadavers examined in the New York Foundling Hospital, found no adenoids present under one year.

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[8] Posey and Wright, Diseases of Nose, Throat, and Ear, vol. ii.

[9] Quoted by Nicholl and Lartigau.

[10] Quoted by Nicholl and Lartigau.

This is the more significant when we consider that the type of child in such an institution is one specially liable to these growths, as shown by the great frequency with which they are found between the third and fourth years.

Heredity seems to be an important factor. In Birkett's series there were fifty-three families with two to four children affected. Tuberculosis, syphilis, gout, alcoholism, and old age in the parents, are mentioned as predisposing causes. The acute infectious fevers are also mentioned as antecedents of importance, but Gradenigo[11] states that although adenoids are supposed to follow these conditions, he has not been able to confirm the view by observation. McBride and Turner point out that such cases have seldom been examined for the presence of adenoids. These may have been present before the illness which has merely aggravated the symptoms and thus drawn attention to them.

Hertoghe[12] came to the conclusion that adenoids are a constant symptom in myxœdema, and that those subjects who show adenoids have at least a predisposition to thyroid disturbance. This has not been accepted, and it has been pointed out that their presence has either been a coincidence or is to be explained on the basis of a common etiology, for adenoids occur commonly in districts where thyroid disturbances are very rare.

A view mentioned by Osler[13] and many American writers, is that the enlargement is probably dependent on a disturbance of the general lymphatic system closely allied to, or a mild type of, lymphatism or status lymphaticus. Ewing has published an interesting paper on this condition and its clinical associations, such as rachitis, various anemias, hemophilia, disturbance of the thyroid, and hypoplasia of various organs. He points out that the common factor of importance appears to be a greatly lowered vital resistance from inherited or acquired influences.

This furnishes a common basis of explanation for the very varied etiology and associated conditions—tuberculosis, syphilis, old age, or other conditions in the parents, or an acute illness in the child, being regarded as acting not in any specific way but merely by lowering the general vitality. Friedländer[14] mentions this view as explaining some of the enlarged lymph nodes met with in childhood in other situations. The local causes are regarded merely as contributory and are the various conditions leading to irritation or congestion. Such are, recurrent attacks of acute inflammation, congenital smallness of the pharynx, or some unhealthy condition in the nasal passage or mouth. Local tuberculosis is regarded by most authorities rather as attacking an adenoid already present than as being a cause of enlargement. In many cases no definite cause can be discovered. In certain cases, contrary to the popular impression, puberty marks a period of increased growth rather than of atrophy.

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[11] Die Hypertrophie der Rachentonsille, Turin, 1901.

[12] Royal Acad. de Med. du Belique, 26 Mar., 1898. (Quoted by Gradenigo.)

[13] Pratice of Medicine.

[14] Arch. Ped., 1902.

THE FUNCTIONS OF THE TONSILLAR TISSUES. The earlier writers confined their notice to the faucial tonsils, which was only natural considering the prominent site of these in the pharynx. There seems to have been a popular belief that they had some obscure relation to the genital apparatus, but this does not seem to have ever been seriously advanced. A more academic view was that they secreted mucus to lubricate the food bolus in its passage down the gullet. As soon as histological studies were made, it was pointed out that they were essentially lymphoid in nature, the mucous glands present being connected with the mucous membrane covering it, not part of the tonsil proper. They resemble very closely in structure the ordinary lymphatic gland or node. This demonstration was important in two ways—it classified the tonsils as part of the general lymphatic system, and it led to the grouping of all the lymphoid collections of the pharynx as part of a whole, namely, the tonsillar ring of Waldeyer. This unity has been shown to be not only histological, but also functional. The function of the tonsillar ring may be conveniently considered from two points of view: (1) As regards the activities arising from the nature of its tissues, and (2) as regards those activities associated with location and environment.

The functions of lymphoid tissues are not wholly understood. They are known to be centres of growth for lymphocytes and hyaline mononuclear lymphocytes (Metchnikoff's macrophages). A better recognized function of the lymph glands or nodes is that of filtration of the lymph. They seem to act as sentinels, stopping, or in any case giving warning, of harmful invasion by the afferent lymphatics. Hingston Fox,[15] in 1885, pointed out that far from being secreting organs, the tonsils are really absorbents, and that their spongy structure penetrated by crypts is designed to give a large absorbing surface.

This absorptive function of the tonsillar ring has been established by experimental and clinical observation. Goodale[16] demonstrated the absorption of carmine particles injected into the tonsillar crypts, and Hendleshon[17] independently showed that similar absorption took place when particles were blown upon the adenoid. Wright[18] found a similar absorption of soot particles in adenoids removed from children that had been living in the smoky atmosphere of Pittsburg. Hodenpyl[19] thought that the tonsils had little power of absorption, but his findings do not agree with those of other observers.

The importance of this demonstration arises from the possibilities it opens up of infections of all kinds arising in the pharynx. Ruffer[20] found that absorption of bacteria went on in the tonsils of healthy animals, the presumption being that they were destroyed without injury to the host. This absorption of bacteria without infection of necessity following, has been recognized as occurring elsewhere, especially in the Peyer's patches, and has modified the views generally held with regard to all infections. The matter has been discussed by Adami[21] under the title "Latent or Subinfection."

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[15] Jour. Anat. and Phys., 1885.

[16] Archiv f. Laryng. and Rhin., Band vii., Heft 2 (Quoted).

[17] Ibid., Band viii., Heft 3 (Quoted).

[18] AMER. JOUR. MED. SCI., 1904.

[19] Ibid., 1891.

[20] Brit. Med. Jour., 1890.

In this connection many authorities, notably Gallois,[22] attribute the constitutional symptoms of adenoids to the absorption of various bacteria or their products from the nasopharynx. Boulay and Heckel[23] make the statement that the cases formerly classified as scrofulous or strumous, may now be divided into three groups: (1) The definitely tuberculous; (2) the syphilitic; and (3) the subjects of adenoids, holding that this latter group will account for all cases not included in the other two. They point out that the degree of *adenoidism* in such cases is not at all proportionate to the size of the growth, but depends entirely on the amount of absorption from it. Their views are worthy of attention and furnish a satisfactory explanation of the rapid and marked improvement so often seen in the patient's general condition after an adenoid operation.

The clinical lesson to be learned from all this is that in all cases of cervical adenitis the nasopharynx should undergo careful examination as a probable source of infection. If an adenoid be present it should be removed as a preliminary to other treatment. It is a common experience to have an adenitis of long standing disappear completely after the removal of adenoids or enlarged tonsils. Many of these cases clinically resemble tuberculous adenitis, but are probably only a prolonged irritation of the glands by continued absorption of the various mouth organisms of low virulence.

In acute general infections the tonsillar ring is now recognized as an important channel, and Ullman[24] has collected a large series of cases in which tonsillitis and sore throat have been followed by general infections, such as articular rheumatism, endocarditis, pleurisy, etc., without any other apparent source of infection.

Goodale[25] has discussed the local pathology of these systemic infections. An invasion by a pathological germ is met in the adenoid or tonsil by a local inflammatory reaction similar to that occurring elsewhere. Systemic infection may take place through the lymphatics or blood stream. Infection is favored by the presence of irregular crypts or pockets resulting from previous inflammation or shrinkage of the tonsil. Goodale[26] has recently emphasized this, and points out that question of removal of such a tonsil is to be decided, not at all by its size, which is the popular criterion, but by the presence of irregularities or recesses interfering with good drainage.

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[21] Jour. Amer. Med. Assoc., December, 1899.

[22] La scrofule et les infections adenoidiennes, second edition, 1905. (Quoted by Boulay and Heckel.[23])

[23] Archiv. Internat. de Laryng. et Otologie, 1906. Nos. 2 and 3.

[24] Med. News, 1901.

[25] Trans. Amer. Laryng. Assoc., 1902.

[26] Boston Med. and Surg. Jour., September 13, 1906.

TUBERCULOSIS OF THE ADENOID AND TONSILLAR TISSUES. The opinion formerly held that the tonsil and adenoid were rarely affected with tuberculosis, has been shown to be incorrect, though as yet statistics differ as to the frequency and importance of their involvement. This change of opinion has resulted from a more careful and more thorough examination of the tissues. As the type of tuberculosis found is usually proliferative, rather than destructive, the clinical and macroscopic examinations are of little value and the methods of diagnosis are histological examination, inoculation of animals, and examination of sections for the tubercle bacilli.

For an indisputable diagnosis all three methods should have a positive result, but, owing to technical difficulty this is often impossible. It is generally accepted that a typical histological picture is sufficient to establish the diagnosis; that the existence of histologically typical tubercles is to be accepted as proof positive of the presence of the disease; and that the converse proves nothing, that is, absence of tubercles does not surely indicate absence of the tubercle bacilli.

Lewin[27] experimented with tuberculin as a method of diagnosis, but found it unsuitable. He got a severe reaction in many cases, in which the adenoid was not found tuberculous after removal. This is explained by the presence of other latent foci, especially in the peribronchial glands.

An important distinction is to be drawn between the two classes of cases of adenoid or tonsillar tuberculosis, the primary, and the secondary. The primary are such as present no sign of tuberculosis elsewhere which might have been the source of infection, and it is with this class of case that I am chiefly concerned.

The frequency of primary adenoid tuberculosis has varied remarkably in the experience of different observers. McBride and Turner, with histological examination, found 3 cases in 100 examined (3 per cent.), but they considered this too low. Dieulafoy,[28] using inoculation, found 7 cases in 35 examined (20 per cent.); Pluder and Fisher[29] with histological examination, found 5 in 32 cases (15.6 per cent.); Nicholl and Lartigau, by histological and inoculation examination, found 12 in 75 cases (16 per cent.). Wood[30] collected 1671 cases, examined by various methods, and found 88 cases of tuberculosis (5.2 per cent.). This corresponds to the work of Lewin, who found 10 in 200 cases (5 per cent.). In the present series of 75 cases, 4 (5.3 per cent.) were regarded as primarily tuberculous.

Primary adenoid tuberculosis is of importance as a latent focus of tuberculosis not recognizable clinically, which may lead to an infection of the cervical glands, or even to a more generalized tuberclosis. The result of operation in the primary cases is favorable in every way. The site of operation heals as usual. The benefit of removing such a focus is apparent, provided always the focus be primary.

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[27] Frankel's Archiv f. Laryng., 1899. (Quoted by Gradenigo.)

[28] Bull. de l'Acad. de Med., Paris, 1895. (Quoted.)

[29] Archiv. f. Laryng. and Rhin, 1896, Band iv. (Quoted.)

[30] Jour. Amer. Med. Assoc., 1904.

The interest lies not so much in the result of any individual case, as in the demonstration of a primary invasion at this point in the respiratory tract. Although opinion has been strong as to the importance of this channel of infection the amount of positive evidence has been small.

Secondary cases are those which arise in persons with pulmonary tuberculosis, or active tuberculosis elsewhere, and which are regarded as infected by the medium of the sputum or blood stream. This is of interest chiefly as demonstrating the readiness with which tubercle bacilli are taken up by the tonsils when present on the surface. Formerly thought uncommon it has been shown to be very frequent. Walsham[31] examined the tonsils in 34 subjects dead of pulmonary tuberculosis; in only 2 had there been anything to attract attention to the tonsils during life, yet in 20 subjects they were found tuberculous. Wood has collected 136 such cases, including those just mentioned, 94 (69 per cent.) being found tuberculous.

From the frequency of these secondary deposits, it is generally held that before a case may be regarded as primary, the patient must remain free from signs of pulmonary tuberculous for at least a year after removal of the tuberculous adenoid. Cases have been reported of patients, previously regarded as healthy, developing signs of pulmonary tuberculosis shortly after removal of the tonsils and adenoids. The explanation usually given of these cases is that an early or latent focus was already present in the lung at the time of operation, though perhaps escaping notice. The anesthetic and shock of the operation in these cases have probably been sufficient to stir up the activity of the disease. Apart from those in the lungs other foci are not likely to be sources of infection for the adenoid, unless a general dissemination with miliary tuberculosis occurs. The presence of a tuberculous focus elsewhere does not exclude an independent primary infection of the adenoid.

ADENOIDS AND TONSILS AS CHANNELS OF GENERAL INFECTION IN TUBERCULOSIS. The importance of the tonsillar ring as a channel of infection in tuberculosis is determined primarily by the relation which these tissues bear to the tuberculosis so commonly present in the cervical glands. Watson Cheyne[32] considered the infrequency of adenoid or tonsillar tuberculosis as strongly against this channel, and quoted the work of Delepine and Younge[33] to support his view. These observers examined the tonsils of 17 cases of tuberculous cervical adenitis, but did not find any of them tuberculous. He pointed out that, if such absorption goes on, the cervical glands should be very often involved in pulmonary tuberculosis in which the tonsils are bathed in sputum more or less laden with tubercle bacilli. He regarded this as uncommon. Walsham, however, has investigated this latter point with a vastly different result. In 27 cases of pulmonary tuberculosis, he found at autopsy evidence of tuberculosis in the cervical glands, 26 times; the one negative case was one of fibroid phthisis. He points out that the impression of their infrequent involvement must have been based on clinical or macroscopic examination.

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[31] Channels of Infection in Tuberculosis. Weber Parkes, Prize Essay, 1903.

[32] Brit. Med. Jour., 1899.

[33] Quoted by Moore.

Moore[34] interprets the negative findings of Delepine and Younge quite differently to Cheyne. He regards the absence of tubercles in the tonsils as a sign of failure on their part to react to the infection. He regards this failure to react as evidence of an abnormal patency or "vulnerability of the channels of infection." He regards the finding of tubercles in the adenoid or tonsil as favorable, in so far as it implies an attempt to check the spread of the infection.

The experimental evidence has been clearly in favor of the tonsillar route. Baup[35] induced an experimental tuberculosis of the cervical glands in rabbits by inoculation of the tonsils, usually without a thoracic or abdominal tuberculosis. Wood produced a similar result in hogs. He states that in his opinion, in 90 per cent. of cases of tuberculous cervical adenitis the channel of entrance has been through the adenoid or tonsils. In so far as the cervical glands are concerned, opinion is fairly uniform as to the importance of this channel of entrance. Of course, it is recognized that other channels, such as carious teeth, are to be remembered, and also the possibility of an ascending infection from the peribronchial and mediastinal glands, but these are considered much less frequent.

When we attempt to estimate the importance of this channel in systemic tuberculosis, we find the matter becoming more dfficult and opinions differing widely. It is now recognized that the tubercle bacillus can be taken up without leaving any trace of its passage at the point of entrance. For this reason the matter cannot be reduced to the form of a definite demonstration; there must always remain a certain amount of inference and therefore possible doubt.

The channels of primary systemic infection, which are regarded as of the most importance, are four, given in the ascending order of importance: (1) By the blood—fœtal infection from a diseased placenta; (2) inoculation—by a wound in the skin; (3) by the digestive tract—following the ingestion of tuberculous food, or of tuberculous material introduced into the mouth by the fingers, especially in young children; and (4) by the respiratory tract—following the inhalation of tubercle bacilli with dust. At present we are only concerned with the last mentioned, namely, inhalation.

The present views as to the mode of infection by inhalation are entirely different to those at first advanced in favor of this route. The idea that the bacilli are inhaled into the lung proper is debated, and the peribronchial glands are now regarded as the primary active focus; it is believed that the lungs or pleural cavity are invaded either by direct extension or by spread along the lymphatics of the lung against the lymph current. The frequency of tuberculosis of the peribronchial glands requires mention only to emphasize it. Northrup[36] in 125 autopsies on tuberculous patients at the New York Foundling Hospital, regarded the infection as primary in the peribronchial glands in 88 cases; in 34 cases the primary site was indeterminate, and in 3 cases in the mesenteric glands. Bovaird[37] collected 75 additional cases from the same source, in 60 of which the primary focus was peribronchial and in 15 indeterminate. It has long been recognized that these nodes are also the common site of latent or obsolete tuberculosis in the adult. Loomis[38] examined the peribronchial nodes of 30 adults dying of acute illness or accident while in apparent robust health, and found that in 8 cases they set up tuberculosis in a rabbit. In these cases there was at autopsy no sign of tuberculosis, past or present.

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[34] Jour. Path. and Bact., 1900.

[35] Les Amygdales Porte d'entree de la Tuberculose, Paris. (Geo. Carre and Co., 1900; quoted by Wood.[30])

The point requiring further study is the channel by which the bacilli reach the peribronchial glands. We may conceive of the penetration of the bacilli occurring at any point between the entrance to the nostrils and the bronchi. Experimentally it has been shown that such infection may occur even from the nasal passages. Renshaw[39] introduced tuberculous material into the noses of guinea-pigs without mechanical injury and was able to produce systemic infection through the cervical glands. In no case was the spread to the meninges.

Boulay and Heckel have declared themselves strongly in favor of the view that this penetration takes place in the nasopharynx. They point out that persons of that type of physique, long regarded as especially predisposing to tuberculosis, having the long narrow chest and the characteristic facies, will generally be found to be the subject of adenoids, past or present. They regard the absorption of the tubercle bacilli as occurring, not especially through the adenoid, but from an unhealthy mucosa anywhere in the nasal passages or pharynx; the adenoid, however, presents conditions particularly favorable to such invasion. They call attention to the increasing frequency with which primary tuberculous conditions have been noted in the nose and nasopharynx since the development of rhinology has brought this region within the scope of clinical diagnosis, and in their opinion the lack of autopsy evidence is due to want of routine examination of the nasal chambers at autopsy. They lay stress on the frequency with which pulmonary tuberculosis is preceded by a history of chronic catarrhal inflammation of the nose and throat, with or without an accompanying cervical adenitis. They point out that in certain individuals pulmonary tuberculosis is preceded by a fairly long premonitory period during which, in their opinion, the infection is confined to the glandular system, the rhinocervicomediastinal chain, and that much may be done at this period by treating any possible channel of entrance in the upper air passages, especially an adenoid. They write, of course, on the hypothesis that the nasopharynx is the main channel of infection in pulmonary tuberculosis. It must be remembered, however, that the conditions of the pharynx they describe are usually associated with catarrhal inflammation in the trachea and bronchi, which renders these regions equally open to invasion if infection be presenst; the exposure to infective material, however, is not so great. In nasal respiration the conditions are such that air-borne particles are not likely to penetrate beyond the nasopharynx. It has been shown experimentally that when a current of air is deflected by a moist surface its bacterial contents are deposited on that surface. The deflecting surface in nasal respiration is the nasopharynx. In mouth breathing, the chances of air-borne infection reaching the trachea or bronchi are somewhat greater. An adenoid or nasopharyngeal catarrh may in this way be an indirect cause of invasion in certain cases.

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[36] New York Med. Jour., February 21, 1891.

[37] Ibid., July 1, 1899.

[38] Medical Record, 1890, vol. ii, p. 689.

[39] Jour. Path. and Bact., 1901, p. 142.

A general dissemination with acute miliary tuberculosis may follow the invasion of the cervical glands, but this is a relatively infrequent event.

CONCLUSIONS. 1. Primary tuberculosis occurs in a certain proportion of all cases of adenoids. From the figures of other observers, and my own, this seems to be about 5 per cent. This is regarded as a conservative estimate.

2. In determining the presence of adenoid tuberculosis the histological method is the most satisfactory.

3. Tuberculosis does not appear to be an important factor in the production of adenoid hypertrophy.

4. Adenoids and tonsils are the important channels of infection in tuberculosis of the cervical glands.

5. In the development of pulmonary tuberculosis adenoids may sometimes be direct channels of infection, but their importance is probably more often indirect by predisposing to catarrhal inflammations of the upper respiratory tract.

I wish to express my thanks to Dr. Birkett for the opportunity of doing this work for his department, and for his constant encouragement during its progress; and to Dr. Adami for the opportunity to work in the pathological department and for his advice in preparing this paper. I must also thank Dr. Keenan for his advice and assistance in the pathological work. The photomicrographs were prepared by Mr. Wootton.